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Differences in mortality and morbidity in African Caribbean and European people with non-insulin dependent diabetes mellitus: results of 20 year follow up of a London cohort of a multinational study

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Abstract

***Objective*—To examine differences in morbidity and mortality due to non-insulin dependent diabetes in African Caribbeans and Europeans.**

***Design*—Cohort study of patients with non-insulin dependent diabetes drawn from diabetes clinics in London. Baseline investigations were performed in 1975-7; follow up continued until 1995.**

***Patients*—150 Europeans and 77 African Caribbeans with non-insulin dependent diabetes.**

***Main outcome measures*—All cause and cardiovascular mortality; prevalence of microvascular and macrovascular complications.**

***Results*—Duration of diabetes was shorter in African Caribbeans, particularly women. African Caribbeans were more likely than the Europeans to have been given a diagnosis after the onset of symptoms and less likely to be taking insulin. Mean cholesterol concentration was lower in African Caribbeans, but blood pressure and body mass index were not different in the two ethnic groups. Prevalence of microvascular and macrovascular complications was insignificantly lower in African Caribbeans than in Europeans. 59 Europeans and 16 African Caribbeans had died by the end of follow up. The risk ratio for all cause mortality was 0.41 (95% confidence interval 0.23 to 0.73) (P = 0.002) for African Caribbeans v Europeans. This was attenuated to 0.59 (0.32 to 1.10) (P = 0.1) after adjustment for sex, smoking, proteinuria, and body mass index. Further adjustment for systolic blood pressure, cholesterol concentration, age, duration of diabetes, and treatment made little difference to the risk ratio. Unadjusted risk ratios for cardiovascular and ischaemic heart disease were 0.33 (0.15 to 0.70) (P = 0.004) and 0.37 (0.16 to 0.85) (P = 0.02) respectively.**

***Conclusions*—African Caribbeans with non-insulin dependent diabetes maintain a low risk of heart disease. Management priorities for diabetes developed in one ethnic group may not necessarily be applicable to other groups.**

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Introduction

People with non-insulin dependent diabetes mellitus continue to be at a greater risk than the general population of dying young, particularly of ischaemic heart disease.[1-4] Morbidity, generally related to the microvascular complications of diabetes, is also high. The avoidance of these complications and the reduction in the risk of excess mortality are prime therapeutic aims of caring for people with diabetes. Management guidelines to achieve these aims are informed by studies that examine relations between risk factors and disease to show where scarce resources should be targeted. But most of these studies have been performed in European populations, with the assumption that the risk factors and their relations are consistent across all ethnic groups. These assumptions may not, however, be true.[5-7] This has important implications for funding health care where people from minority ethnic groups constitute a substantial proportion of the diabetic population.

Studies examining mortality and complication rates in minority ethnic groups are limited and have generally been performed in the United States, where access to health care is highly inequitable,[8 9] potentially confounding comparisons of complication rates and mortality by ethnic group. To our knowledge, no study determining ethnic differences in mortality has been completed in the United Kingdom, where access to appropriate health care is less likely to be a key factor in determining the burden of disease and subsequent risk of death. We examined the morbidity and mortality associated with diabetes in a 20 year follow up study of African Caribbeans and Europeans with non-insulin dependent diabetes in the United Kingdom.

Subjects and methods

Data from the London cohort of the World Health Organisation's multinational study of vascular disease in diabetes were used for these analyses. Detailed methods

for this study have been published elsewhere.[10 11] In brief, 14 centres around the world took part in the original cross sectional survey during 1975-7. People with diabetes who were aged 35-55 were recruited from clinic lists or population based surveys. At that time there were no internationally agreed criteria for the diagnosis of diabetes, which was generally diagnosed on clinical grounds supported by measurement of blood glucose concentration. London was one of the participating centres, and all patients attending the outpatient clinics of King's College, Guy's, and St Thomas's Hospitals at least once over a one year period formed the sampling frame. This was to ensure that the sample was not biased towards frequent attenders at the clinics. These patients were then stratified by age (35-41, 42-48, and 49-55 years), duration of diabetes (1-6, 7-13, and ≥14 years), and sex, creating 18 cells. This stratification was designed to achieve comparable samples across all WHO centres. The aim was to examine 28 people per cell, and to achieve this 35 people were selected at random from each of these cells and invited to participate in the study, either at their next clinic visit or by post. Of those who were asked, 90% agreed to participate and 85% were eventually examined. These London clinics serve inner city areas that include a comparatively large proportion of African Caribbeans—that is, people of black African descent who either were born in the Caribbean or were descendants of those born in the Caribbean. Current clinic based studies of people with diabetes, particularly non-insulin dependent diabetes, may not be representative of patients with diabetes as many patients are now wholly managed in general practice. However, this was certainly not the case in the mid-1970s, when very few people with diabetes escaped the attentions of a hospital clinic.

Insulin dependent diabetes was defined as that which required continuous treatment with insulin within one year of diagnosis. The remainder were regarded as having non-insulin dependent diabetes.

At baseline, participants were asked to complete a questionnaire that included details of medical history, mode of diagnosis (from symptoms or on screening), current treatment, and health related behaviours. Investigations included measurement of height (in metres) and weight (in kilograms) without outdoor clothes or shoes, and resting blood pressure was measured with a standard mercury sphygmomanometer. Funduscopy was performed through dilated pupils, and the lesions observed were compared with standard photographs. The degree of retinopathy in the worse eye was used to assign a retinopathy category. A resting 12 lead electrocardiogram was taken and classified according to the Minnesota code.[12] Non-fasting blood samples were taken for measurement of total cholesterol and serum creatinine concentrations. These were both measured at the WHO collaborating laboratory in Atlanta (USA). A fresh sample of urine was provided to test for proteinuria with the salicylsulphonic acid test. Ethnic group was assigned by the investigator on the basis of appearance and country of birth. Country of birth was a valid proxy of ethnic group in this age group at that time as migration from the Caribbean to the United Kingdom began only in the 1950s[13]; thus all our participants were first generation migrants. At baseline 95 European men, 55 European women, 27 African Caribbean men, and 50 African Caribbean women with non-insulin dependent diabetes were examined.

A second morbidity examination was performed in 1983, when subjects were asked to attend for a follow up investigation which included a questionnaire and physical examination. The prevalence of complications at this follow up was calculated by assuming that all those who reattended who had had a given complication at baseline had that complication at follow up; this number was added to the number of new complications that had developed since the baseline examination.

All patients were tagged by supplying the Office of Population Censuses and Surveys' central registry with their NHS number, name, address, and date of birth to see whether they had died or emigrated. Only deaths up to 1 January 1995 were included in these analyses. When possible, data from hospital records or post-mortem reports were collected to provide additional information about the cause of death. A mortality panel was convened to assign underlying cause of death. Mortality analyses were performed for all causes of death, circulatory disease (*International Classification of Diseases*, ninth revision (ICD-9) codes 390-459 or 798), and ischaemic heart disease (codes 410-414).

In all analyses smoking was classified as never smoker, ex-smoker, and current smoker. Retinopathy was defined as one or more red lesions; as hard or soft exudates in either eye or vitreous opacity; or as haemorrhage or new vessel formation. Proteinuria was defined as slight turbidity or worse in the salicylsulphonic acid test. Prevalent ischaemic heart disease was defined as either a doctor diagnosed history of ischaemic heart disease or infarction or major Q waves (Minnesota code 1-1 or 1-2) on electrocardiography.[12]

STATISTICAL ANALYSES

Mean values for continuous variables and percentages for categorical variables at baseline were calculated separately by sex and ethnic group. Differences were compared between ethnic groups for both sexes. Median blood pressure was calculated by ranking all blood pressures and assigning treated hypertension the highest rank.[14] This method is preferable to calculating mean blood pressures as it addresses the problems of variation in treatment rates for hypertension between ethnic groups and in the efficacy of antihypertensive treatment. We also adjusted for age for continuous variables and performed age standardisation of categorical variables, but as the results did not differ from the unadjusted values we present the unadjusted values.

Kaplan-Meier curves were plotted to examine survival times by ethnic group. Differences in survival times were tested for significance by the log rank test. Further analyses were performed using Cox's proportional hazards modelling to examine the modifying effects of other risk factors on mortality. The proportionality assumption was tested by adding a term for the interaction of time with the explanatory variable of interest to the basic model, in other words, by including a time dependent variable. If the regression parameter for this time dependent variable is significantly different from zero, the relative hazard is dependent on time and thus challenges the null hypothesis of no change in the hazard ratio with time.[15] These tests showed that the proportionality assumptions were not violated. Variables that were thought to influence mortality were fitted separately with ethnic group, and the effect of these on the maximum likelihood estimates were compared. Variables that seemed to be important were fitted together and their combined effects were then compared to determine whether any variables ceased to be of importance and could be discarded. Other variables that did not seem to be important on their own were also fitted in these combined models to check that they were not important in the presence of others.[16] Interactions between important covariates were also tested. Sex of participants was forced into the final model as there are clear sex differences in mortality within ethnic group and, in this study, ethnic differences in the sex balance of the sample. The final models presented in the tables include only the participants who did not have any missing data on any of the variables included in the models.

Table 1—*Baseline data for European and African Caribbean people with non-insulin dependent diabetes. Values are numbers (percentages) of patients or means unless stated otherwise*

	Men			Women		
	European (n = 95)	African Caribbean (n = 27)	P value	European (n = 55)	African Caribbean (n = 50)	P value
Age (years)	48	47	0.6	48	48	0.7
Duration of diabetes (years)	7	6	0.2	9	6	0.04
Age at onset (years)	41	42	0.6	39	42	0.06
Body mass index (kg/m^2)	26.3	25.1	0.1	28.2	27.9	0.8
Median blood pressure (mm Hg):						
Systolic	136	128	0.8	138	138	0.9
Diastolic	88	86	0.2	88	90	0.1
Cholesterol (mmol/l)	5.9	5.4	0.07	6.1	5.3	0.001
Current smokers	33 (35)	12 (43)	0.5	23 (42)	11 (22)	0.02
Creatinine (mmol/l)	1.00	1.10	0.01	0.80	0.87	0.03
Diagnosis from:						
Symptoms	63 (66)	19 (70)	0.4	32 (58)	40 (80)	0.02
Routine screening	32 (34)	8 (30)	0.8	20 (36)	8 (16)	0.02
Treatment:						
Insulin only	22 (23)	2 (7)	0.07	17 (31)	3 (6)	0.001
Oral agents only	44 (46)	17 (63)	0.1	29 (53)	39 (78)	0.007
Heart disease	12 (13)	1 (4)	0.2	2 (4)	1 (2)	0.6
Retinopathy	26 (27)	6 (22)	0.6	19 (35)	15 (30)	0.5
Proteinuria	21 (22)	2 (8)	0.1	13 (24)	8 (16)	0.3

Both age and duration of diabetes may be strong determinants of the risk of dying prematurely and may be more appropriate terms to define time than simple person years of follow up. Modelling was therefore repeated using age and duration of diabetes as the time variable rather than person years of follow up. The SAS and EGRET statistical packages were used for all analyses.[17 18]

Results

At baseline the duration of diabetes was shorter—significantly so in women—for African Caribbeans (6 years) compared with Europeans (9 years, P = 0.04) (table 1). Age at onset of diabetes was also higher in African Caribbeans, significantly so for women. Compatible with this was the finding that in women the diagnosis of diabetes in African Caribbeans was more likely after the onset of symptoms than during routine screening. In contrast, African Caribbeans of both sexes were less likely to be treated with insulin and more likely to be receiving oral treatment. Median systolic and diastolic blood pressure and mean body mass index were not different in the two ethnic groups, but mean serum cholesterol concentration was lower in African Caribbeans than Europeans. The percentage of current smokers was particularly low in African Caribbean women (21%) compared with European women (42%, P = 0.02).

The prevalence of microvascular and macrovascular complications at baseline was lower, but not significantly so, in African Caribbeans compared with Europeans. At the second morbidity examination in 1983 there was no ethnic difference in the prevalence of any of the microvascular complications, but the prevalence of coronary heart disease was 10% (11/109) in Europeans and 2% (1/65) in African Caribbeans (P = 0.03). Of those who had survived until the second examination, 17% (22/131) of Europeans and 11% (8/74) of African Caribbeans did not attend. Non-attendance rates were compared by complications at baseline. Any retinopathy was used as the indicator variable for complications at baseline as this complication had a prevalence at baseline high enough to split the sample evenly. Of those without retinopathy at baseline, 10% (11/105) of Europeans and 9% (5/56) of African Caribbeans did not attend. Of those with retinopathy at baseline, 24% of both Europeans (11/45) and African Caribbeans (5/21) did not attend. Therefore, although attendance at the follow up examination was biased towards those who were comparatively free of complications at baseline, this bias operated equally in both ethnic groups.

At the end of the follow up period 59 Europeans (39%) and 16 African Caribbeans (21%) had died. The median follow up was 18 person years (range 0-20), with a total of 3498 person years of follow up. During this time seven Europeans and 10 African Caribbeans emigrated from the United Kingdom.

The risk of death from any cause was substantially lower in African Caribbeans than Europeans (fig 1; log rank statistic = 9.55, P = 0.002). These differences persisted when the mortality risk ratios were adjusted for other risk factors (table 2). African Caribbeans had less than half the risk of death from all causes compared with the Europeans (0.41 (95% confidence interval 0.23 to 0.73), P = 0.002). Body mass index, proteinuria, and smoking seemed to be important predictors of mortality in separate models. But the ethnic difference in mortality persisted after adjustment for sex, body mass index, proteinuria, and smoking (0.59 (0.32 to 1.10)), although the hazard ratio became non-significant (P = 0.1). The addition of other potential confounders into the model, such as cholesterol concentration, systolic blood pressure, age, duration of diabetes, and type of treatment, did not significantly alter the risk ratio. Mortality from circulatory disease (risk ratio 0.33, P = 0.004) and ischaemic heart disease (risk ratio 0.37, P = 0.02) was similarly lower in African Caribbeans compared with Europeans. Again, these risks were attenuated when other risk factors were adjusted for, the greatest attenuation occurring when body mass index and proteinuria were added to the models. There was no evidence of an interaction between ethnic group and either body mass index or proteinuria on the risk of death.[12]

The reanalyses for all cause mortality with age as the time variable produced an unadjusted risk ratio of 0.48 (P = 0.01) for African Caribbeans compared with Europeans. When duration of diabetes was substituted as the time variable, this risk ratio became 0.55 (P = 0.04). Adjustment for the variables used in previous models did not qualitatively alter these risk ratios, but as before risk ratios became non-significant in the final model.

Discussion

People of black African descent worldwide enjoy a certain protection from heart disease, despite high rates of diabetes.[19-23] Whether this protection persists in people with diabetes is conflicting. Data from the United States indicate that among people with diabetes

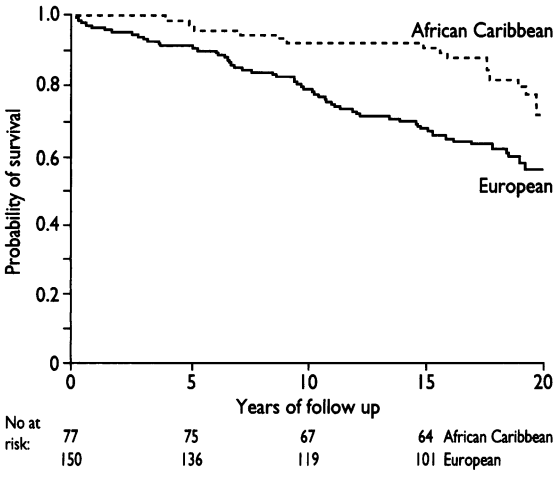

Fig 1—*Survival of African Caribbeans and Europeans with non-insulin dependent diabetes mellitus*

Table 2—*Rate ratios (95% confidence intervals) for mortality from all causes, circulatory disease, and ischaemic heart disease in African Caribbeans compared with Europeans with diabetes*

Cause of death	No of deaths	No of participants	Hazard ratio (95% confidence interval)	P value
All causes				
Europeans	59	150		
African Caribbeans	16	77		
African Caribbeans *v* Europeans				
Unadjusted			0.41 (0.23 to 0.73)	0.002
Adjusted for:				
Sex			0.42 (0.24 to 0.76)	0.004
+Body mass index			0.47 (0.26 to 0.85)	0.01
+Proteinuria			0.56 (0.31 to 1.02)	0.06
+Smoking			0.59 (0.32 to 1.10)	0.1
Circulatory disease				
Europeans	39	150		
African Caribbeans	9	77		
African Caribbeans *v* Europeans				
Unadjusted			0.33 (0.15 to 0.70)	0.004
Adjusted for:				
Sex			0.35 (0.16 to 0.77)	0.009
+Body mass index			0.45 (0.20 to 1.01)	0.05
+Proteinuria			0.54 (0.24 to 1.24)	0.1
+Smoking			0.54 (0.23 to 1.24)	0.1
Ischaemic heart disease				
Europeans	31	150		
African Caribbeans	8	77		
African Caribbeans *v* Europeans				
Unadjusted			0.37 (0.16 to 0.85)	0.02
Adjusted for:				
Sex			0.40 (0.17 to 0.92)	0.03
+Body mass index			0.51 (0.22 to 1.22)	0.1
+Proteinuria			0.63 (0.26 to 1.53)	0.3
+Smoking			0.64 (0.26 to 1.58)	0.3

African Americans have similar or slightly lower death rates compared with white Americans.[4 24] Also, diabetes does not increase the risk of cardiovascular mortality in African Americans to the same extent as it does in white Americans.[25] More strikingly though, African Americans with diabetic end stage renal disease survived twice as long as their white counterparts.[26]

Data from the United Kingdom are equally sparse. Routine mortality statistics show that African Caribbeans, both those in the general population and those with diabetes recorded on the death certificate, have about half to three quarters the risk of dying of heart disease of Europeans.[19 27] But information from these routine data may be biased differently by ethnic group as comparisons are dependent on the reporting of diabetes[28] and other causes of death on the certificate, and the classification of ethnic group is based solely on country of birth. A cohort study overcomes most of these limitations, and examination of the London component of the WHO study provides a unique opportunity to determine ethnic differences in the risk of dying prematurely. We found that total and cardiovascular mortality in African Caribbeans was about half that in Europeans. This halving in risk persists but becomes non-significant after adjustment for other factors that are generally thought to influence mortality.

We also confirmed that the prevalence of heart disease in African Caribbeans with diabetes is lower than that in Europeans, both at baseline and at the follow up examination.[29 30] The reasons for this protection from heart disease are not entirely clear from our study. Like others, we found little ethnic difference in obesity (as measured by body mass index) and systolic blood pressure,[29-31] in contrast with studies of the general population.[32] This reflects findings in the United States, where the difference in systolic blood pressure between African Americans and white Americans is almost negligible in those with diabetes in comparison with the non-diabetic population.[24] The lower rates of cigarette smoking, particularly in African Caribbean women,[29] could not account for the low risk of mortality from heart disease.

POSSIBLE REASONS FOR LOW HEART RISK IN AFRICAN CARIBBEANS

A striking finding from this and other studies is the comparatively low prevalence of previous heart disease in people of black African descent.[24 26] This runs counter to the general understanding of the relative frequencies of non-insulin dependent diabetes and heart disease within populations, in that both are thought to be consequences of insulin resistance, resulting in glucose intolerance on the one hand and lipid disturbances on the other.[33] One proposal is that the diabetic population in those of black African descent consists of a mixture of insulin sensitive and insulin resistant people.[34] Thus, although insulin concentrations are higher in African Caribbeans than Europeans in the general population[32] and such increases are associated with a raised prevalence of glucose intolerance, lipid patterns are generally highly favourable.[6 7 29 35] We measured only cholesterol concentration, but the results were favourable in this diabetic population of African Caribbeans.

Obesity may be the key to understanding the lack of association between glucose intolerance and lipid patterns in African Caribbeans. Although body mass index is not different in African Caribbean and European men in the general population, central obesity, as assessed by the waist to thigh ratio, is much lower in African Caribbeans.[7] Central obesity is associated with cardiovascular morbidity and mortality,[36] so that populations with a low degree of central obesity may enjoy a certain protection from heart disease.

ETHNIC DIFFERENCES IN MICROVASCULAR COMPLICATIONS

We did not find clear ethnic differences in microvascular complications.[29] The high serum creatinine concentrations found in this and other studies[29] probably reflect the greater muscle mass of African Caribbeans. This contrasts with the results of other studies showing that severe microvascular complications—that is, blindness and end stage renal disease—are much more common in African Americans than in white Americans.[24] This, however, may be due to the inequitable access to health care that has been consistently observed in African Americans in the United States.[37-39] This degree of inequitable access to care is not thought to occur in the United Kingdom, with the "free at the point of delivery" NHS, and this may therefore account for the equivalent complication rates observed in the United Kingdom. Support for this comes from studies in the United States. Amputation rates in those with diabetes are consistently higher in African Americans than white Americans,[40] but when access to health care is not based on ability to pay amputation rates are equal in the two ethnic groups.[41]

LIMITATIONS OF DATA

Our study has several limitations. The numbers of people, particularly of African Caribbeans, are small and cannot support full analyses separately by sex. Despite this, we found important ethnic differences in both risk factors for disease and mortality. Data on important risk factors for mortality, such as measures of diabetes control, central obesity, and triglyceride and high density lipoprotein cholesterol concentrations were not collected. However, our limited data on lipids reflect findings from other studies. Despite these shortcomings, ours is one of the few cohort studies of people with diabetes that starts to explore ethnic differences in mortality, with some adjustment for confounders.

CONCLUSIONS

We conclude that African Caribbeans with diabetes maintain their protection from heart disease and that this protection may be due to a low degree of central obesity, resulting in a reduced atherogenic lipid profile. Further investigations are required to disentangle these complex relations.

Key messages

- Populations with high rates of diabetes tend to have high rates of heart disease
- People of black African descent worldwide have low rates of heart disease despite having high rates of diabetes
- This study found that this protection from heart disease persists in people with diabetes: African Caribbeans with diabetes have a third of the risk of dying from heart disease compared with Europeans
- Assumptions about risk factor relations derived from studies in European populations may not necessarily hold true for other ethnic groups
- Establishing the reasons for this relative protection may further the understanding of heart disease and may provide valuable clues to help reduce the risk of heart disease in all populations

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ONE HUNDRED YEARS AGO

THE SUMMER HOLIDAYS.

ALTHOUGH the lapse of time is not without its sadness, perhaps at no season does its aspect present less sadness than during the summer holidays. At this season the physician sees his patients turn their backs upon him without regret even with a secret satisfaction; nay it is within the bound of dreams that the patient may be pleased to get rid of his doctor. Of course he soon finds out his mistake, but that is not altogether bad for him. *O fortunati nimium sua si bona norint!*

How then shall we hint at holidays to the patient who may see too much of us; and how meanwhile may we make the best of the chance he may give us of our own escape? With the greater facility for moving about it is getting more and more difficult to get a holiday. Whether we think first of the patient or of ourselves, we realise with no little dismay that to go to one of the great London terminuses to make for a main route, whether to a British or foreign health resort, is to be swept into a stampede of buffaloes, with the exception, that buffaloes arrive and we do not. Nor is it otherwise when we are ultimately deposited somewhere or other. If we go abroad we find ourselves insignificant members of a crowd, probably consisting chiefly of middle-class Germans and their families; excellent fellows whose birthdays return every night at dinner time, and who—thank goodness!—go off about six o'clock every morning in droves, with bouquets tied to their walking staves. By midday they are back again, and for the next few hours they talk a good deal, smoke more, and cough and so forth more still. (*BMJ* 1896;ii:518.)